Malignant Ventricular Arrhythmias as the Initial Manifestation of Hyperthyroidism

To the Editor:

Ventricular arrhythmia is normally secondary to organic heart disease or ion disorders. On rare occasions, it may also be caused by extracardiac diseases, such as hyperthyroidism. We describe a 46-year-old woman who consulted for dyspnea, cough, and edema that had lasted three weeks. She reported no other symptoms and had not lost weight. Soon after her arrival, she presented cardiorespiratory arrest caused by ventricular fibrillation, but recovered with electric defibrillation.

The subsequent physical examination showed a patient with anxiety and slight fine tremor in both hands. She presented dyspnea at rest, crepitate rales in both bases, jugular vein enlargement, and edema in the legs. Blood pressure was 155/64 mm Hg. Auscultation revealed a systolic murmur in the mitral area and gallop rhythm in the third sound. The electrocardiogram basically showed sinus rhythm of 100 bpm, normal QRS complex, and QTc interval of 0.44 s. The chest x-ray disclosed cardiomegaly and bilateral interstitial pattern indicative of edema. The general blood tests were normal (potassium, 3.8 mmol/L; magnesium, 1.8 mg/dL). The echocardiogram showed left ventricle neither dilated nor hypertrophied, with global hypokinesia (ejection fraction, 35%), and mild to moderate mitral insufficiency.

In the first 7 hours of hospitalization, she presented repeated runs of polymorphic ventricular tachycardia in torsade de pointes (Figure 1) that partially remitted with magnesium sulfate and virtually disappeared after initiating atenolol therapy at 72 h post-admission. Cardiac catheterization did not show any coronary lesions or vasospasm. The thyroid hormone study revealed the following: T3 of 733.1 ng/dL (normal value, 60-181 ng/dL), T4 of 20.2 µg/dL (normal value, 4.5-10.9 µg/dL), free T4 of 5.3 ng/dL (normal value, 0.8-1.8 ng/dL), thyroid-stimulating hormone (TSH) <0.01 mU/L (normal value, 0.3-5.5 mU/L), antithyroglobulin antibodies <20.0 U/mL (normal value <40 U/mL), and antimicrosomal antibodies 2,704 U/mL (normal value <35 U/mL). Thyroid scintigraphy showed hyperfunctioning diffuse goiter. Therefore, Graves-Basedow disease was diagnosed, and carbimazole therapy was started, maintaining the atenolol therapy. The QTc interval was 0.42 s. An echocardiogram at 14 days showed an improvement in systolic function (ejection fraction, 53%) and reduced mitral regurgitation. At the end of follow-up (8 March 8, 2006), the patient was well and presented no signs of heart failure or arrhythmias.

The most common clinical manifestations of thyrotoxic heart disease are heart rate disorders, in particular, sinus tachycardia and atrial fibrillation, which presents in 5%-15% of patients. Malignant ventricular arrhythmias, which are potentially fatal, are much more unusual. The onset of tachycardia or ventricular fibrillation has been reported within a thyrotoxic storm. The presentation of these arrhythmias in the initial phase of the disease is much less common, and only a few isolated cases are described in the scientific literature. The majority occur in the context of thyrotoxic periodic paralysis with severe hypopotassemia. There has been an occasional patient in whom the ventricular arrhythmia were related to coronary spasm. An interesting observation is the finding of QT interval prolongation, in both experimental work and in patients with hyperthyroidism, which could play an important role in the pathophysiological mechanism of ventricular tachycardia in torsade de pointes.

In short, the patient we describe is an illustration of a rare arrhythmic complication of hyperthyroidism. Ventricular dysfunction attributable to the hyperthyroidism and mild hypopotassemia have probably contributed to the genesis of the ventricular arrhythmia. Although exceptionally rare, hyperthyroidism should be investigated and ruled out in patients who present ventricular arrhythmia of uncertain etiology.

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Figure 1. Polymorphic ventricular tachycardia in torsade de pointes.
REFERENCES


LETTERS TO THE EDITOR

Pheochromocytoma presents with headache, sweating, and palpitations associated with hypertension, heart failure, and arrhythmias due to catecholamine secretion. The presence of sustained ventricular tachycardia (VT) has been reported rarely. We describe a case of pheochromocytoma that manifested as sustained and incessant monomorphic VT.

A 47-year-old woman with diabetes and hypertension under therapy, who had experienced episodes of back pain, palpitations, and dyspnea from 5 months earlier, was seen in the emergency room during an episode. Blood pressure was 130/65 mm Hg and troponin T 0.30 ng/mL (normal <0.01), with normal creatine kinase and myoglobin. The baseline electrocardiogram was normal (Figure 1). However, during follow-up, the patient presented sustained and incessant runs of ordinary tachycardia with a QRS complex of 0.11 s at 180 bpm, pattern of right bundle-branch block and inferior axis, and atrioventricular dissociation (Figure 2), that reproduced the symptoms. The echocardiography showed left ventricular concentric hypertrophy with normal systolic function; the coronary angiography was normal. Based on the typical pattern of left fascicular origin, intravenous and oral verapamil was administered, stabilizing the sinus rhythm. Monitoring during hospitalization showed episodes of atrial tachycardia with a narrow QRS complex, with heart rates around 150 bpm and persistent sinus tachycardia. Syncopal symptoms were associated with a systolic blood pressure of 240 mm Hg, which dropped to 70 mm Hg within a few minutes without treatment. Thyroid hormone and TSH concentrations were normal, but urinary metanephrines were high. Computed tomography showed a right adrenal mass with a diameter of 6 cm; metaiodobenzylguanidine scintigraphy showed uptake by the mass. Phenoxybenzamine and atenolol therapy stabilized the blood pressure and prevented recurrence of ventricular and atrial tachycardia. A unilateral adrenalectomy was performed, with the diagnosis of pheochromocytoma confirmed by histology. Following surgery, the hypertension was controlled and urinary metanephrines were normalized. Two years after surgery, the patient remained asymptomatic, with no recurrence of arrhythmia and with normal blood pressure.

The most common cardiologic manifestations of pheochromocytoma are heart failure and myocarditis or dilated cardiomyopathy. Ventricular tachycardia has been described in case studies, and there are no data on the mechanism of this tachycardia, although torsade de pointes has occasionally been reported in relation to a long QT interval. In our case, the QT interval was normal and the tachycardia pattern indicated a fascicular origin, but the association with atrial tachycardia pointed to a systemic arrhythmogenic factor. Elevated catecholamines may cause abnormal sinoatrial node pacemaker activity and/or triggering focal activity, and the suppression of pheochromocytoma arrhythmias with alpha and beta blockers has been described. After the diagnosis of pheochromocytoma, the patient was treated with phenoxybenzamine and atenolol, which stabilized the blood pressure and prevented recurrence of ventricular and atrial tachycardia. A unilateral adrenalectomy was performed, with the diagnosis of pheochromocytoma confirmed by histology. Following surgery, the hypertension was controlled and urinary metanephrines were normalized. Two years after surgery, the patient remained asymptomatic, with no recurrence of arrhythmia and with normal blood pressure.